Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	2527	screen same depression	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:41
L2	14	l1 same agent	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:50
L3	2527	screen same depression	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:42
L4	0	l2 and 800/18.ccls.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:50
L5	175	800/18.ccls. and depression	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:51
L6	144	l5 and screen	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:51
L7	133	l6 and behavior$	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:51
L8	65	l7 not allen.in.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:52
L9	128	l7 not wisotzkey.in.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:52
L10	60	l8 not wisotzkey.in.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:52
L11	62	l8 not deltagen.as.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:54
L12	60	l10 not leviten.as.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:55
L13	45	l10 not leviten.in.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:55
L14	44	l13 not phillips.in.	US-PGPUB; USPAT	OR	OFF	2004/12/21 12:57
L15	14	l14 and secreted	US-PGPUB; USPAT	OR	OFF	2004/12/21 14:25
L16	0	screted adj protein adj gene	US-PGPUB; USPAT	OR	OFF	2004/12/21 14:26
L17	91	secreted adj protein adj gene	US-PGPUB; USPAT	OR	OFF	2004/12/21 14:26
L18	3	l17 and 800/8.ccls.	US-PGPUB; USPAT	OR	OFF	2004/12/21 14:27
L19	1	l17 and 800/18.ccls.	US-PGPUB; USPAT	OR	OFF	2004/12/21 14:27
L20	1	l17 and 800/14.ccls.	US-PGPUB; USPAT	OR	OFF	2004/12/21 14:27

(FILE 'HOME' ENTERED AT 09:05:04 ON 21 DEC 2004)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, LIFESCI' ENTERED AT 09:05:36 ON 21 DEC 2004

L1 0 S SECRETED (A) PROTIEN
L2 25413 S SECRETED (A) PROTEIN
L3 249 S L2 AND KNOCKOUT
L4 1 S L2 (W) KNOCKOUT
L5 132 DUP REM L3 (117 DUPLICATES REMOVED)
L6 39 S L5 NOT PY>2000

=> d bib ab 16 17 20 24 25 29

L6 ANSWER 16 OF 39 MEDLINE on STN
AN 1999413979 MEDLINE
DN PubMed ID: 10484471
TI Collagen accumulation is decreased in SPARC-null mice with bleomycin-induced pulmonary fibrosis.
AU Strandjord T P; Madtes D K; Weiss D J; Sage E H
CS Department of Pediatrics, University of Washington, Seattle, WA 98195-6320, USA.. tps@u.washington.edu
NC GM-40711 (NIGMS)
HL-03017 (NHLBI)
HL-49401 (NHLBI)
SO American journal of physiology, (1999 Sep) 277 (3 Pt 1) L628-35. Journal code: 0370511. ISSN: 0002-9513.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
ED Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991028
AB **Secreted protein** acidic and rich in cysteine (SPARC) has been shown to be coexpressed with type I collagen in tissues undergoing remodeling and wound repair. We speculated that SPARC is required for the accumulation of collagen in lung injury and that its absence would attenuate collagen accumulation. Accordingly, we have assessed levels of collagen in SPARC-null mice in an intratracheal bleomycin-injury model of pulmonary fibrosis. Eight- to ten-week-old SPARC-null and wild-type (WT) mice received bleomycin (0.0035 U/g) or saline intratracheally and were subsequently killed after 14 days. Relative levels of SPARC mRNA were increased 2.7-fold ($P < 0.001$) in bleomycin-treated WT lungs in comparison with saline-treated lungs. Protein from bleomycin-treated WT lung contained significantly more hydroxyproline (191.9 microg/lung) than protein from either bleomycin-treated SPARC-null lungs or saline-treated WT and SPARC-null lungs (147.4 microg/lung, 125.4 microg/lung, and 113. 0 microg/lung, respectively; $P < 0.03$). These results indicate that SPARC is increased in response to lung injury and that accumulation of collagen, as indicated by hydroxyproline content, is attenuated in the absence of SPARC. The properties of SPARC as a matricellular protein associated with cell proliferation and matrix turnover are consistent with its participation in the development of pulmonary fibrosis.

L6 ANSWER 17 OF 39 MEDLINE on STN
AN 1999308170 MEDLINE
DN PubMed ID: 10379362
TI Uteroglobin: a novel cytokine?.
AU Mukherjee A B; Kundu G C; Mantile-Selvaggi G; Yuan C J; Mandal A K; Chattopadhyay S; Zheng F; Pattabiraman N; Zhang Z
CS Section on Developmental Genetics, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland

20892-1830, USA.. mukherja@exchange.nih.gov
SO Cellular and molecular life sciences : CMLS, (1999 May) 55 (5) 771-87.
Ref: 179
Journal code: 9705402. ISSN: 1420-682X.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199907
ED Entered STN: 19990715
Last Updated on STN: 19990715
Entered Medline: 19990706
AB Blastokinin or uteroglobin (UG) is a steroid-inducible, evolutionarily conserved, multifunctional **protein secreted** by the mucosal epithelial of virtually all mammals. It is present in the blood and in other body fluids including urine. An antigen immunoreactive to UG antibody is also detectable in the mucosal epithelia of all vertebrates. UG-binding proteins (putative receptor), expressed on several normal and cancer cell types, have been characterized. The human UG gene is mapped to chromosome 11q12.2 13.1, a region that is frequently rearranged or deleted in many cancers. The generation of UG **knockout** mice revealed that disruption of this gene causes: (i) severe renal disease due to an abnormal deposition of fibronectin and collagen in the glomeruli; (ii) predisposition to a high incidence of malignancies; and (iii) a lack of polychlorinated biphenyl binding and increased oxygen toxicity in the lungs. The mechanism(s) of UG action is likely to be even more complex as it also functions via a putative receptor-mediated pathway that has not yet been clearly defined. Molecular characterization of the UG receptor and signal transduction via this receptor pathway may show that this protein belongs to a novel cytokine/chemokine family.

L6 ANSWER 20 OF 39 MEDLINE on STN
AN 1999199293 MEDLINE
DN PubMed ID: 10097146
TI Loss of transformed phenotype in cancer cells by overexpression of the uteroglobin gene.
AU Zhang Z; Kundu G C; Panda D; Mandal A K; Mantile-Selvaggi G; Peri A; Yuan C J; Mukherjee A B
CS Section on Developmental Genetics, Heritable Disorders Branch, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD 20892-1830, USA.
SO Proceedings of the National Academy of Sciences of the United States of America, (1999 Mar 30) 96 (7) 3963-8.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199905
ED Entered STN: 19990525
Last Updated on STN: 19990525
Entered Medline: 19990512
AB Uteroglobin (UG) is a multifunctional, **secreted protein** that has receptor-mediated functions. The human UG (hUG) gene is mapped to chromosome 11q12.2-13.1, a region frequently rearranged or deleted in many cancers. Although high levels of hUG expression are characteristic of the mucosal epithelia of many organs, hUG expression is either drastically reduced or totally absent in adenocarcinomas and in viral-transformed epithelial cells derived from the same organs. In agreement with these findings, in an ongoing study to evaluate the effects of aging on UG-**knockout** mice, 16/16 animals developed malignant tumors, whereas the wild-type littermates (n = 25) remained apparently

healthy even after 11/2 years. In the present investigation, we sought to determine the effects of induced-expression of hUG in human cancer cells by transfecting several cell lines derived from adenocarcinomas of various organs with an hUG-cDNA construct. We demonstrate that induced hUG expression reverses at least two of the most important characteristics of the transformed phenotype (i.e., anchorage-independent growth on soft agar and extracellular matrix invasion) of only those cancer cells that also express the hUG receptor. Similarly, treatment of the nontransfected, receptor-positive adenocarcinoma cells with purified recombinant hUG yielded identical results. Taken together, these data define receptor-mediated, autocrine and paracrine pathways through which hUG reverses the transformed phenotype of cancer cells and consequently, may have tumor suppressor-like effects.

L6 ANSWER 24 OF 39 MEDLINE on STN
AN 1999072597 MEDLINE
DN PubMed ID: 9856777
TI SPARC deficiency leads to early-onset cataractogenesis.
AU Norose K; Clark J I; Syed N A; Basu A; Heber-Katz E; Sage E H; Howe C C
CS The Wistar Institute, Philadelphia, Pennsylvania 19104, USA.
NC EY04542 (NEI)
GM40711 (NIGMS)
SO Investigative ophthalmology & visual science, (1998 Dec) 39 (13) 2674-80.
Journal code: 7703701. ISSN: 0146-0404.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199812
ED Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981221
AB PURPOSE: To determine the role of SPARC (**secreted protein**, acidic, and rich in cysteine) in cataractogenesis by examining mice deficient in a matricellular protein SPARC. METHODS: Mice were rendered SPARC-deficient by a targeted disruption of the gene. Slit-lamp microscopy and histology were used to examine the eyes of SPARC-null and wild-type mice from birth to 14 months of age. RESULTS: SPARC-null mice developed opacities in the posterior cortex of the eye as early as 1.5 months after birth. The diffuse cataracts appeared to progress toward the anterior cortex and reached maturity in many animals by 3.5 months of age. Early stages of cataractogenesis in SPARC-null mice included inhibition of normal lens fiber cell differentiation, degeneration of fiber cells, vacuole formation at the equator, and liquefaction of the cortex. No cataracts were detected in wild-type mice up to the age of 8 months. CONCLUSIONS: The early onset of cataracts in SPARC-null mice establishes that the gene is essential to the maintenance of lens transparency.

L6 ANSWER 25 OF 39 MEDLINE on STN
AN 1999072353 MEDLINE
DN PubMed ID: 9856533
TI Uteroglobin: physiological role in normal glomerular function uncovered by targeted disruption of the uteroglobin gene in mice.
CM Comment in: Am J Kidney Dis. 1998 Dec;32(6):1084-5. PubMed ID: 9856529
Comment in: Am J Kidney Dis. 2000 Feb;35(2):362-3. PubMed ID: 10676744
AU Mukherjee A B; Kundu G C; Mandal A K; Pattabiraman N; Yuan C J; Zhang Z
CS Section on Developmental Genetics, Heritable Disorders Branch, The National Institute of Child Health and Human Development, The National Institutes of Health, Bethesda, MD 20892-1830, USA..
mukherja@exchange.nih.gov
SO American journal of kidney diseases : official journal of the National Kidney Foundation, (1998 Dec) 32 (6) 1106-20. Ref: 151
Journal code: 8110075. ISSN: 1523-6838.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199901
ED Entered STN: 19990115
Last Updated on STN: 20010521
Entered Medline: 19990104
AB Blastokinin or uteroglobin (UG) is an evolutionarily conserved, steroid-inducible, homodimeric, multifunctional, **secreted protein** with potent Immunomodulatory/antiinflammatory properties. Recently, a UG-receptor expressed on several malignant and normal cell types has been characterized. Although the biochemistry, structural, and molecular biology of UG have been extensively studied, its physiological function(s), until recently, remained unknown. By generating UG-null (UG-/-) mice, we determined that an essential role of UG is to prevent severe renal disease caused by an abnormal deposition of predominantly multimeric fibronectin (Fn) and collagen in the glomerulus. The molecular mechanisms by which UG prevents this disease in control (UG+/+) mice, at least in part, is attributable to its high-affinity binding to Fn and the formation of Fn-UG heteromers, which counteract both Fn-Fn and Fn-collagen interactions, required for abnormal tissue deposition. In addition, by inhibiting secretory phospholipase A2 (sPLA2) activity and decreasing the level of lysophosphatidic acid (LPA), UG may indirectly prevent the activation of integrins (eg, alpha5beta1) that enhance abnormal tissue deposition of Fn. The mechanism(s) of UG action is likely to be even more complex, because it also functions through a receptor-mediated pathway that has not yet been clearly defined. Nevertheless, the UG gene-**knockout** mice provide a valuable animal model for investigation of human glomerulopathies in general and familial Fn-deposit glomerulopathy in particular.

L6 ANSWER 29 OF 39 MEDLINE on STN
AN 1998241484 MEDLINE
DN PubMed ID: 9573043
TI osteoprotegerin-deficient mice develop early onset osteoporosis and arterial calcification.
AU Bucay N; Sarosi I; Dunstan C R; Morony S; Tarpley J; Capparelli C; Scully S; Tan H L; Xu W; Lacey D L; Boyle W J; Simonet W S
CS Department of Molecular Genetics, Amgen, Inc., Thousand Oaks, California 91320-1789, USA.
SO Genes & development, (1998 May 1) 12 (9) 1260-8.
Journal code: 8711660. ISSN: 0890-9369.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199806
ED Entered STN: 19980611
Last Updated on STN: 19980611
Entered Medline: 19980601
AB Osteoprotegerin (OPG) is a **secreted protein** that inhibits osteoclast formation. In this study the physiological role of OPG is investigated by generating OPG-deficient mice. Adolescent and adult OPG-/- mice exhibit a decrease in total bone density characterized by severe trabecular and cortical bone porosity, marked thinning of the parietal bones of the skull, and a high incidence of fractures. These findings demonstrate that OPG is a critical regulator of postnatal bone mass. Unexpectedly, OPG-deficient mice also exhibit medial calcification of the aorta and renal arteries, suggesting that regulation of OPG, its signaling pathway, or its ligand(s) may play a role in the long observed association between osteoporosis and vascular calcification.